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FILE 'MEDLINE, EMBASE, BIOSIS' ENTERED AT 17:50:39 ON 08 DEC 2004
L1          3168 S MCEWAN?/AU OR MCKERNAN?/AU
L2         23863 S "TWO HYBRID" OR "2 HYBRID" OR "INTERACTION TRAP SYSTEM"
L3         11377 S "GENOME WIDE" OR "PROTEOME WIDE" OR "PROTEIN INTERACTION MAP"
L4         55014 S "CDNA LIBRARY" OR "DNA GENOMIC LIBRARY" OR "GENOMIC LIBRARY"
L5          3841 S HYPERMETHYLATED OR HYPOMETHYLATED OR (METHIONINE (S) ENRICHED
L6             4 S BACTERIOMATCH
L7         10266 S "PROTEIN MAPPING" OR "PROTEIN MAP"
L8         16945 S "96" (S) WELL
L9           945 S "384" (S) WELL
L10        15407 S ("10,000" OR "1,000,000" OR 1,000?) (S) CELL?
L11            7 S L1 AND L2
L12            3 DUP REM L11 (4 DUPLICATES REMOVED)
L13            1 S L10 AND L5
L14            3 S L10 AND L2
L15            3 DUP REM L14 (0 DUPLICATES REMOVED)
L16           14 S L10 AND L3
L17            9 DUP REM L16 (5 DUPLICATES REMOVED)
L18          957 S "PROTEIN ARRAY"
L19            4 S L18 AND L10
L20            3 DUP REM L19 (1 DUPLICATE REMOVED)
L21          120 S L7 AND L2
L22            4 DUP REM L6 (0 DUPLICATES REMOVED)
L23           10 S L21 AND L4
L24            5 DUP REM L23 (5 DUPLICATES REMOVED)
L25            0 S L21 AND L5
L26           50 S L5 AND L3
L27           19 DUP REM L26 (31 DUPLICATES REMOVED)
L28            1 S L27 AND "REVIEW"
L29          252 S BAIT (P) PREY
L30          282 S BAIT AND PREY
L31          476 S 30 AND L2
L32            4 S L31 AND L3
L33           51 S L31 AND L4
L34            0 S L33 AND L18
L35           30 DUP REM L33 (21 DUPLICATES REMOVED)
L36            2 DUP REM L32 (2 DUPLICATES REMOVED)
L37          957 S "PROTEIN ARRAY"
L38        83394 S "PROTEIN INTERACTION"
L39           50 S L37 AND L38
L40            5 S L39 AND L2
L41            5 DUP REM L40 (0 DUPLICATES REMOVED)
L42          305 S (L8 OR L9) AND (MICROARRAY OR ARRAY)
L43            2 S L42 AND L38
L44            2 DUP REM L43 (0 DUPLICATES REMOVED)
L45         6263 S L38 AND L2
L46          537 S L45 AND  "REPORTER"
L47           46 S L46 AND (L3 OR L4)
L48           36 DUP REM L47 (10 DUPLICATES REMOVED)
L49           28 S L48 NOT PY>=2003
L50          346 S "PROTEIN-PROTEIN" (S) MAP
L51           66 S L50 AND L2
L52           39 DUP REM L51 (27 DUPLICATES REMOVED)

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ANSWER 1 OF 5 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
ACCESSION NUMBER: 2002:431982 BIOSIS
DOCUMENT NUMBER: PREV200200431982
TITLE: A novel pancreas Regeneration-(Reg-) binding protein.
AUTHOR(S): Zhang, Hong [Reprint author]; Wu, Haiyan; Fan, Zuoheng; Patel, Sameer; Mueller, Cathy M. [Reprint author]; Zenilman, Michael E. [Reprint author]
CORPORATE SOURCE: Surgery, SUNY-Downstate Medical Center, 450 Clarkson Ave., Brooklyn, NY, 11203, USA
SOURCE: FASEB Journal, (March 22, 2002) Vol. 16, No. 5, pp. A1154. print.
Meeting Info.: Annual Meeting of Professional Research Scientists on Experimental Biology. New Orleans, Louisiana, USA. April 20-24, 2002.
CODEN: FAJOEC. ISSN: 0892-6638.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 14 Aug 2002
Last Updated on STN: 14 Aug 2002

AB Pancreas Regeneration (Reg) protein is secretary protein from acinar cells. It can stimulate ductal and islet beta-cell growth. In diabetic animal models, Reg I improves glucose tolerance. To study the mechanism of Reg I intracellular signaling, we employed the yeast-**2-hybrid** system in ARIP, a rat ductal cell line which responds to Reg. The rat Reg coding sequence was cloned into the pBD-GAL4 Cam (bait) vector, and a **cDNA library** of the target cell ARIP was cloned in to the vector pAD-GAL4-2.1 (target). The clones were transformed into YRG-2 yeast, and assayed expression of LacZ reporter gene by the detection of beta-galactosidase activity. LacZ+ clone were isolated and sequenced. A homology search revealed that the entire DNA sequence of mitogen activating kinase phosphatase-1 (MKP-1) gene. We also demonstrated the association of Reg I with MKP-1 by co-immunoprecipitation. MKP-1 belongs to a class of dual-specificity phosphatase able to reverse the activation of mitogen-activated **protein** (**MAP**) kinase family member and its function has been related to insulin gene regulation. We propose that pancreas Reg I protein exerts its mitogenic effect in ductal and beta cells through the MKP pathway.

L24 ANSWER 2 OF 5 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2002280109 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12020817
TITLE: HIV-1 integrase interacts with yeast microtubule-associated proteins.
AUTHOR: de Soultrait Vaea Richard; Caumont Anne; Durrens Pascal; Calmels Christina; Parissi Vincent; Recordon Patricia; Bon Elisabeth; Desjobert Cecile; Tarrago-Litvak Laura; Fournier Michel
CORPORATE SOURCE: UMR 5097 CNRS-Universite Victor Segalen Bordeaux 2, BP 103, Bat. 3A-3 Etage, 146 rue Leo Saignat, 33076 Bordeaux X Cedex, France.
SOURCE: Biochimica et biophysica acta, (2002 May 3) 1575 (1-3) 40-8.
Journal code: 0217513. ISSN: 0006-3002.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200207
ENTRY DATE: Entered STN: 20020522
Last Updated on STN: 20020720

Entered Medline: 20020719

AB The human immunodeficiency virus type 1 (HIV-1) integrase (IN) mediates the insertion of viral DNA into the human genome. In addition to IN, cellular and viral proteins are associated to proviral DNA in the so-called preintegration complex (PIC). We previously reported that the expression of HIV-1 IN in yeast leads to the emergence of a lethal phenotype. This effect may be linked to the IN activity on infected human cells where integration requires the cleavage of genomic DNA. To isolate and characterize potential cellular partners of HIV-1 IN, we used it as a bait in a **two-hybrid** system with a yeast **genomic library**. IN interacted with proteins belonging to the microtubule network, or involved in the protein synthesis apparatus. We focused our interest on one of the selected inserts, L2, which corresponds to the C-end half of the yeast STU2p, a microtubule-associated **protein** (**MAP**). STU2p is an essential component of the yeast spindle pole body (SPB), which is able to bind microtubules in vitro. After expressing and purifying L2 as a recombinant protein, we showed its binding to IN by ELISA immunodetection. L2 was also able to inhibit IN activity in vitro. In addition, the effect of L2 was tested using the "lethal yeast phenotype". The coexpression of IN and the L2 peptide abolished the lethal phenotype, thus showing important in vivo interactions between IN and L2. The identification of components of the microtubule network associated with IN suggest a role of this complex in the transport of HIV-1 IN present in the PIC to the nucleus, as already described for other human viruses.

L24 ANSWER 3 OF 5 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

ACCESSION NUMBER: 2002177965 EMBASE

TITLE: HIV-1 integrase interacts with yeast microtubule-associated proteins.

AUTHOR: De Soultrait V.R.; Caumont A.; Durrens P.; Calmels C.; Parissi V.; Recordon P.; Bon E.; Desjobert C.; Tarrago-Litvak L.; Fournier M.

CORPORATE SOURCE: M. Fournier, UMR 5097 CNRS-Univ. V. S. Bordeaux 2, Bat. 3A-3 Etage, 146 rue Leo Saignat, 33076 Bordeaux Cedex, France. Michel.Fournier@reger.u-bordeaux2.fr

SOURCE: Biochimica et Biophysica Acta - Gene Structure and Expression, (3 May 2002) 1575/1-3 (40-48).
Refs: 43
ISSN: 0167-4781 CODEN: BBGSD5

PUBLISHER IDENT.: S 0167-4781(02)00241-5

COUNTRY: Netherlands

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 004 Microbiology

LANGUAGE: English

SUMMARY LANGUAGE: English

AB The human immunodeficiency virus type 1 (HIV-1) integrase (IN) mediates the insertion of viral DNA into the human genome. In addition to IN, cellular and viral proteins are associated to proviral DNA in the so-called preintegration complex (PIC). We previously reported that the expression of HIV-1 IN in yeast leads to the emergence of a lethal phenotype. This effect may be linked to the IN activity on infected human cells where integration requires the cleavage of genomic DNA. To isolate and characterize potential cellular partners of HIV-1 IN, we used it as a bait in a **two-hybrid** system with a yeast **genomic library**. IN interacted with proteins belonging to the microtubule network, or involved in the protein synthesis apparatus. We focused our interest on one of the selected inserts, L2, which corresponds to the C-end half of the yeast STU2p, a microtubule-associated **protein** (**MAP**). STU2p is an essential component of the yeast spindle pole body (SPB), which is able to

bind microtubules in vitro. After expressing and purifying L2 as a recombinant protein, we showed its binding to IN by ELISA immunodetection. L2 was also able to inhibit IN activity in vitro. In addition, the effect of L2 was tested using the "lethal yeast phenotype". The coexpression of IN and the L2 peptide abolished the lethal phenotype, thus showing important in vivo interactions between IN and L2. The identification of components of the microtubule network associated with IN suggest a role of this complex in the transport of HIV-1 IN present in the PIC to the nucleus, as already described for other human viruses.

L24 ANSWER 4 OF 5 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2001315015 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11278711
TITLE: A novel tobacco mitogen-activated **protein** (**MAP**) kinase kinase, NtMEK1, activates the cell cycle-regulated p43Ntf6 MAP kinase.
AUTHOR: Calderini O; Glab N; Bergounioux C; Heberle-Bors E; Wilson C
CORPORATE SOURCE: Institute of Microbiology and Genetics, University of Vienna, Dr. Bohrgasse 9, A-1030 Vienna, Austria.
SOURCE: Journal of biological chemistry, (2001 May 25) 276 (21) 18139-45.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AJ302651
ENTRY MONTH: 200107
ENTRY DATE: Entered STN: 20010709
Last Updated on STN: 20030105
Entered Medline: 20010705

AB **Two-hybrid** screening of a tobacco BY-2 cell suspension **cDNA library** using the p43(Ntf6) mitogen-activated **protein** (**MAP**) kinase as bait resulted in the isolation of a cDNA encoding a protein with features characteristic of a MAP kinase kinase (MEK), which has been called NtMEK1. **Two-hybrid** interaction analysis and pull-down experiments showed a physical interaction between NtMEK1 and the tobacco MAP kinases p43(Ntf6) and p45(Ntf4), but not p43(Ntf3). In kinase assays NtMEK1 preferentially phosphorylated p43(Ntf6). Functional studies in yeast showed that p43(Ntf6) could complement the yeast MAP kinase mutant mpk1 when co-expressed with NtMEK1, and that this complementation depended on the kinase activity of p43(Ntf6). Expression analysis showed that the NtMEK1 and ntf6 genes are co-expressed both in plant tissues and following the induction of cell division in leaf pieces. These data suggest that NtMEK1 is an MEK for the p43(Ntf6) MAP kinase.

L24 ANSWER 5 OF 5 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 1998250765 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9582351
TITLE: Regulation of protein phosphatase 2A activity by caspase-3 during apoptosis.
AUTHOR: Santoro M F; Annand R R; Robertson M M; Peng Y W; Brady M J; Mankovich J A; Hackett M C; Ghayur T; Walter G; Wong W W; Giegel D A
CORPORATE SOURCE: Department of Biochemistry, Parke-Davis Pharmaceutical Research Division, Warner-Lambert Company, Ann Arbor, Michigan 48105, USA.
SOURCE: Journal of biological chemistry, (1998 May 22) 273 (21) 13119-28.

Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199806
ENTRY DATE: Entered STN: 19980708
Last Updated on STN: 20000303
Entered Medline: 19980625

AB Although the available evidence suggests that whereas the caspase family plays a major role in apoptosis, they are not the sole stimulators of death. A random yeast **two-hybrid** screen of a lymphocyte **cDNA library** (using caspase-3 as the bait) found an interaction between caspase-3 and the regulatory subunit Aalpha of protein phosphatase 2A. This protein was found to be a substrate for caspase-3, but not caspase-1, and could compete effectively against either a protein or synthetic peptide substrate. In Jurkat cells induced to undergo apoptosis with anti-Fas antibody, protein phosphatase 2A (PP2A) activity increased 4.5-fold after 6 h. By 12 h, the regulatory Aalpha subunit could no longer be detected in cell lysates. There was no change in the amount of the catalytic subunit. The effects on PP2A could be prevented by the caspase family inhibitors acetyl-Asp-Glu-Val-Asp (DEVD) aldehyde or Ac-DEVD fluoromethyl ketone. The mitogen-activated **protein** (**MAP**) kinase pathway is regulated by PP2A. At 12 h after the addition of anti-Fas antibody, a decrease in the amount of the phosphorylated forms of MAP kinase was observed. Again, this loss of activated MAP kinase could be prevented by the addition of DEVD-cho or DEVD-fmk. These data are consistent with a pathway whereby induction of apoptosis activates caspase-3. This enzyme then cleaves the regulatory Aalpha subunit of PP2A, increasing its activity. These data show that the activated PP2A will then effect a change in the phosphorylation state of the cell. These data provide a link between the caspases and signal transduction pathways.

NSWER 1 OF 1 MEDLINE on STN
ACCESSION NUMBER: 1998290846 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9625817
TITLE: DNA methylation in urological malignancies (**review**).
AUTHOR: Schulz W A
CORPORATE SOURCE: Urologische Klinik, Heinrich-Heine-Universitat, D-40225 Dusseldorf, Germany.
SOURCE: International journal of oncology, (1998 Jul) 13 (1) 151-67. Ref: 308
Journal code: 9306042. ISSN: 1019-6439.
PUB. COUNTRY: Greece
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199808
ENTRY DATE: Entered STN: 19980828
Last Updated on STN: 19980828
Entered Medline: 19980820

AB Three different kinds of alterations in DNA methylation have been observed in urological malignancies. DNA hypermethylation of CpG-rich promoter regions is an important mechanism involved in the inactivation of tumor suppressor and other genes in prostate, renal cell, and bladder carcinoma. **Genome-wide** hypomethylation is most pronounced in urothelial carcinoma, but also occurs in prostatic cancer. Loss of imprinting may be a primary event in the aetiogenesis of Wilms' tumor and probably contributes to testicular cancer. With respect to alterations in DNA methylation three tumor categories are distinguished: in the development of embryonic tumors, e.g. Wilms' tumor, loss of imprinting is important probably by upsetting the balance between genes promoting or inhibiting proliferation. In tumors with faulty DNA methylation, e.g. renal cell carcinoma, occasional errors in DNA methylation are selected for during tumor development. In tumors with deranged methylation, e. g. in most bladder and prostate carcinomas, the mechanisms establishing methylation patterns are fundamentally disturbed and multiple alterations in DNA methylation are observed. At least one of the enzymes establishing methylation patterns, viz. DNA methyltransferases and demethylases, may be deregulated. Moreover, changes in methyl group metabolism need to be considered. DNA hypermethylation and loss of imprinting act by altering the expression of selected genes, whereas hypomethylation may facilitate transcription and recombination throughout the genome by its effect on the chromatin structure. The combination of all three types of alterations may create genomic instability in tumors with deranged DNA methylation. Regarding a potential clinical use, detection of hypermethylation appears most promising in cancer diagnosis, while parameters reflecting **genome-wide** hypomethylation may prove useful in the prediction of prognosis. Inhibitors of DNA methylation are being improved and will presumably first be employed against tumors with **hypermethylated** key tumor suppressor genes.

ANSWER 2 OF 2 MEDLINE on STN DUPLICATE 1

ACCESSION NUMBER: 2001545194 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11559592
TITLE: A protein-**protein interaction** **map** of the Caenorhabditis elegans 26S proteasome.
AUTHOR: Davy A; Bello P; Thierry-Mieg N; Vaglio P; Hitti J; Doucette-Stamm L; Thierry-Mieg D; Reboul J; Boulton S; Walhout A J; Coux O; Vidal M
CORPORATE SOURCE: CRBM, CNRS UPR-1086, IFR 24, 34293 Montpellier, France.
CONTRACT NUMBER: 232 (NHGRI)
5R01HG01715-02 (NCI)
7R33 CA81658-02 (NCI)
P01CA80111-02
SOURCE: EMBO reports, (2001 Sep) 2 (9) 821-8.
Journal code: 100963049. ISSN: 1469-221X.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200112
ENTRY DATE: Entered STN: 20011011
Last Updated on STN: 20020122
Entered Medline: 20011207

AB The ubiquitin-proteasome proteolytic pathway is pivotal in most biological processes. Despite a great level of information available for the eukaryotic 26S proteasome-the protease responsible for the degradation of ubiquitylated proteins-several structural and functional questions remain unanswered. To gain more insight into the assembly and function of the metazoan 26S proteasome, a **two-hybrid**-based **protein interaction map** was generated using **30** Caenorhabditis elegans proteasome subunits. The results recapitulate interactions reported for other organisms and reveal new potential interactions both within the 19S regulatory complex and between the 19S and 20S subcomplexes. Moreover, novel potential proteasome interactors were identified, including an E3 ubiquitin ligase, transcription factors, chaperone proteins and other proteins not yet functionally annotated. By providing a wealth of novel biological hypotheses, this interaction map constitutes a framework for further analysis of the ubiquitin-proteasome pathway in a multicellular organism amenable to both classical genetics and functional genomics.

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ANSWER 1 OF 5 MEDLINE on STN
ACCESSION NUMBER: 2004399358 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15290761
TITLE: Discovering protein-protein interactions.
AUTHOR: Ng See-Kiong; Tan Soon-Heng
CORPORATE SOURCE: Knowledge Discovery Department, Institute for Infocomm Research, 21 Heng Mui Keng Terrace, Singapore 119613, Singapore.. skng@i2r.a-star.edu.sg
SOURCE: J Bioinform Comput Biol, (2004 Jan) 1 (4) 711-41. Journal code: 101187344. ISSN: 0219-7200.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200409
ENTRY DATE: Entered STN: 20040812
Last Updated on STN: 20040921
Entered Medline: 20040917

AB The ongoing genomics and proteomics efforts have helped identify many new genes and proteins in living organisms. However, simply knowing the existence of genes and proteins does not tell us much about the biological processes in which they participate. Many major biological processes are controlled by **protein interaction** networks. A comprehensive description of protein-protein interactions is therefore necessary to understand the genetic program of life. In this tutorial, we provide an overview of the various current high-throughput methods for discovering protein-protein interactions, covering both the conventional experimental methods and new computational approaches.

L41 ANSWER 2 OF 5 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
ACCESSION NUMBER: 2004:348210 BIOSIS
DOCUMENT NUMBER: PREV200400349961
TITLE: Peptide aptamers: Specific inhibitors of protein function.
AUTHOR(S): Hoppe-Seyler, Felix [Reprint Author]; Crnkovic-Mertens, Irena; Tomai, Evangelia; Butz, Karin
CORPORATE SOURCE: Programm Infekt and Krebs, Deutsch Krebsforschungszentrum, Neuenheimer Feld 242, D-69120, Heidelberg, Germany hoppe-seyler@dkfz.de
SOURCE: Current Molecular Medicine (Hilversum), (August 2004) Vol. 4, No. 5, pp. 529-538. print. ISSN: 1566-5240 (ISSN print).
DOCUMENT TYPE: Article
General Review; (Literature Review)
LANGUAGE: English
ENTRY DATE: Entered STN: 18 Aug 2004
Last Updated on STN: 18 Aug 2004

L41 ANSWER 3 OF 5 MEDLINE on STN
ACCESSION NUMBER: 2003465567 MEDLINE
DOCUMENT NUMBER: PubMed ID: 14527327
TITLE: Proteomics.
AUTHOR: Zhu Heng; Bilgin Metin; Snyder Michael
CORPORATE SOURCE: Department of Molecular, Cellular, and Developmental Biology, Yale University, New Haven, Connecticut 06520, USA.. heng.zhu@yale.edu
SOURCE: Annual review of biochemistry, (2003) 72 783-812. Ref: 100 Journal code: 2985150R. ISSN: 0066-4154.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LANGUAGE: English

FILE SEGMENT: Priority Journals
ENTRY MONTH: 200312
ENTRY DATE: Entered STN: 20031008
Last Updated on STN: 20031218
Entered Medline: 20031209

AB Fueled by ever-growing DNA sequence information, proteomics-the large scale analysis of proteins-has become one of the most important disciplines for characterizing gene function, for building functional linkages between protein molecules, and for providing insight into the mechanisms of biological processes in a high-throughput mode. It is now possible to examine the expression of more than 1000 proteins using mass spectrometry technology coupled with various separation methods. High-throughput yeast **two-hybrid** approaches and analysis of protein complexes using affinity tag purification have yielded valuable protein-**protein interaction** maps. Large-scale protein tagging and subcellular localization projects have provided considerable information about protein function. Finally, recent developments in protein microarray technology provide a versatile tool to study protein-protein, protein-nucleic acid, protein-lipid, enzyme-substrate, and protein-drug interactions. Other types of microarrays, though not fully developed, also show great potential in diagnostics, protein profiling, and drug identification and validation. This review discusses high-throughput technologies for proteome analysis and their applications. Also discussed are the approaches used for the integrated analysis of the voluminous sets of data generated by proteome analysis conducted on a global scale.

L41 ANSWER 4 OF 5 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 2000070588 EMBASE
TITLE: A comprehensive analysis of protein-protein interactions in Saccharomyces cerevisiae.
AUTHOR: Uetz P.; Giot L.; Cagney G.; Mansfield T.A.; Judson R.S.; Knight J.R.; Lockshon D.; Narayan V.; Srinivasan M.; Pochart P.; Qureshi-Emili A.; Li Y.; Godwin B.; Conover D.; Kalbfleisch T.; Vijayadamodar G.; Yang M.; Johnston M.; Fields S.; Rothberg J.M.
CORPORATE SOURCE: J.M. Rothberg, CuraGen Corporation, 555 Long Wharf Drive, New Haven, CT 06511, United States. jrothberg@curagen.com
SOURCE: Nature, (10 Feb 2000) 403/6770 (623-627).
Refs: 41
ISSN: 0028-0836 CODEN: NATUAS
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Two large-scale yeast **two-hybrid** screens were undertaken to identify protein-protein interactions between full-length open reading frames predicted from the Saccharomyces cerevisiae genome sequence. In one approach, we constructed a **protein array** of about 6,000 yeast transformants, with each transformant expressing one of the open reading frames as a fusion to an activation-domain. This array was screened by a simple and automated procedure for 192 yeast proteins, with positive responses identified by their positions in the array. In a second approach, we pooled cells expressing one of about 6,000 activation domain fusions to generate a library. We used a high-throughput screening procedure to screen nearly all of the 6,000 predicted yeast proteins, expressed as Gal4 DNA-binding domain fusion proteins, against the library, and characterized positives by sequence analysis. These approaches resulted in the detection of 957 putative interactions involving 1,004 S. cerevisiae proteins. These data reveal

interactions that place functionally unclassified proteins in a biological context, interactions between proteins involved in the same biological function, and interactions that link biological functions together into larger cellular processes. The results of these screens are shown here.

L41 ANSWER 5 OF 5 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
ACCESSION NUMBER: 2000:430423 BIOSIS
DOCUMENT NUMBER: PREV200000430423
TITLE: Genome-wide **protein interaction** maps using **two-hybrid** systems.
AUTHOR(S): Legrain, Pierre [Reprint author]; Selig, Luc
CORPORATE SOURCE: Hybrigenics, 180 Avenue Daumesnil, Paris, 75012, France
SOURCE: FEBS Letters, (25 August, 2000) Vol. 480, No. 1, pp. 32-36. print.
CODEN: FEBLAL. ISSN: 0014-5793.
DOCUMENT TYPE: Article
General Review; (Literature Review)
LANGUAGE: English
ENTRY DATE: Entered STN: 11 Oct 2000
Last Updated on STN: 10 Jan 2002

AB Automated sequence technology has rendered functional biology amenable to genomic scale analysis. Among genome-wide exploratory approaches, the **two-hybrid** system in yeast (Y2H) has outranked other techniques because it is the system of choice to detect protein-protein interactions. Deciphering the cascade of binding events in a whole cell helps define signal transduction and metabolic pathways or enzymatic complexes. The function of proteins is eventually attributed through whole cell **protein interaction** maps where totally unknown proteins are partnered with fully annotated proteins belonging to the same functional category. Since its first description in the late 1980's, several versions of the Y2H have been developed in order to overcome the major limitations of the system, namely false positives and false negatives. Optimized versions have been recently applied at multi-molecular and genomic scale. These genome-wide surveys can be methodologically divided into two types of approaches: one either tests combinations of predefined polypeptides (the so-called matrix approach) using various short-cuts to speed up the process, or one screens with a given polypeptide (bait) for potential partners (preys) present in complex libraries of genomic or complementary DNA (library screening). In the former strategy, one tests what one knows, for example pair-wise interactions between full-length open reading frames from recently sequenced and annotated genomes. Although based on a one-by-one scheme, this method is reported to be amenable to large-scale genomics thanks to multicloning strategies and to the use of small robotics workstations. In the latter, highly complex cDNA or genomic libraries of protein domains can be screened to saturation with high-throughput screening systems allowing the discovery of yet unidentified proteins. Both approaches have strengths and drawbacks that will be discussed here. None yields a full proteome-wide screening since certain proteins (e.g. some transcription factors) are not usable in Y2H. Novel **two-hybrid** assays have been recently described in bacteria. Applications of these time- and cost-effective assays to genomic screening will be discussed and compared to the Y2H technology.

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PALM INTRANET

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mckernan	k	Search

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